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Review

Scope of medicinal flora as effective anti ulcer agents

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Gastric ulcer generally results from persistent erosions and damage of the stomach wall which become perforated and develops into peritonitis and massive haemorrhage as a result of inhibition of the synthesis of mucus, bicarbonate and prostaglandins. The commercially available antiulcer drugs usually have various side effects. Due to these side effects, there is a need to find new antiulcerogenic compound(s) with potentially less or no side effects and medicinal plants have always been the main source of new drugs for the treatment of gastric ulcer. Several hundred plant genera are used medicinally mainly in the form of herbal preparation in indigenous system of medicine in different countries for the treatment of ulcers. In this review, we have given a detailed description of ulcer and its causative factors along with medicinal plants with antiulcer potency.

Key words: *Helicobacter pylori*, Meckel's Diverticulum ulcer, gastroesophageal reflux disease, peptic ulcer disease.

INTRODUCTION

The human stomach contains enumerable muscles that carry out the process of digestion and switch the different forms of food into digestive fluids which are pepsin and hydrochloric acid. These fluids make the food digest in the stomach. In order to attain this several organs integrate with each other which includes the central nervous system (CNS) and hormonal systems. The ulcer in the stomach may be the result of the disparity in the digestive fluids (Hoogerwerf, 2001). The over production of pepsin or hydrochloric acid may harm the line up of the stomach and cause ulcers in the stomach. Every year 4 million people are diagnosed with this disease. An estimated 6000 people die every year because of the complications associated with stomach ulcer. 40, 000 people undergo surgery in order to get relief from the persistent symptoms of ulcer annually. An estimated 15,000 deaths occur as consequence of PUD. Indian pharmaceutical industry antacid and anti ulcer drugs share 6.2 billion rupees and occupy 4.3% in the market share (Jaikumar, 2010). Two main approaches for treating peptic ulcer include: reducing the production of gastric fluid and re-enforcing gastric mucosal protection (Valle, 2005).

Ulcers are defined as a breach in the mucosa of the alimentary tract, which extends through the muscularis mucosa into the submucosa or deeper (mayoclinic.com, 2011). Ulcus pepticum or PUD or peptic ulcer disease, is a chronic and most often solitary lesion that can be defined as mucosal erosions which is equal to or greater than 0.5 cm of an area of the gastrointestinal tract (mainly stomach and duodenum) which is usually acidic and extremely painful (Vakil, 2010; Chan, 2010).

Types of ulcers include, gastric ulcer (stomach), duodenal ulcer (duodenum), oesophageal ulcer (oesophagus) and meckel's diverticulum ulcer (meckel's diverticulum) (Podein, 2007).

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Signs and symptoms

Abdominal pain, naturally epigastric with severity relating to mealtimes is usually after 1 h of taking a meal. Duodenal ulcers are classically relieved by food, while gastric ulcers are exacerbated by it; bloating and abdominal fullness; water brash (rush of saliva after an episode of regurgitation to dilute the acid in oesophagus); nausea, copious vomiting; loss of apetite and weight loss; hematemesis (vomiting of blood); melena (tarry, foul-smelling feces due to oxidized iron from hemoglobin); heart burn, gastroesophageal reflux disease (GERD) and use of certain forms of medication can raise the suspicion of peptic ulcer; and sudden increase in the abdominal pain or sharpness in the quality of the pain; vomiting blood or material that looks like coffee grounds; blood in stool or black, tarry stools (pharmacology2000.com, 2011) .

A gastric ulcer will give epigastric pain during the meal as gastric acid is secreted, or after the meal, as the alkaline duodenal contents reflux into the stomach. Symptoms of duodenal ulcers will manifest mostly before the meal-when acid production stimulated by hunger is passed into the duodenum. This is not considered as reliable sign in clinical practice (Berroteran, 2002; Medina, 2010).

Helicobacter pylori

Helicobacter pylori was discovered in 1982 by two Australian Scientists, J. Robin Warren and Barry J. Marshall as a causative factor of ulcers. They showed that most stomach ulcers and gastritis were caused by colonization with this bacterium and not by stress or spicy food as had been assumed before. *H. pylori* is a spiral shaped Gram negative bacterium that lives in the acidic environment of the stomach. It is found in stomach along with acid secretion and can damage the tissue of the stomach and duodenum, causing inflammation and ulcers (Tiwari, 2005). *H. pylori* is believed to be transmitted from person to person through the oral cavity. The hypothesis that the mouth is a reservoir for *H. pylori* and a potential source of gastric infection is strengthened by several reports of *H. pylori* DNA in the saliva and dental plaque (Ahmed, 2006; Li, 1995; Olivier, 2006).

Other cause of ulcers is notably the gastric cancer. This especially occurs in ulcers of the greater curvature of the stomach in which most of them are a consequence of chronic *H. pylori* infection (Gloria, 2011; Saluja, 2002; Satoskar, 2007). It has been reported that *H. pylori* infection is a common problem in diabetic patients who have inadequate metabolic control where these micro organisms colonize the gastric antrum (Bikha, 2010). As diabetes and *H. pylori* are considered as major causes for dyspepsia, the incidence of *H. pylori* infection is higher in diabetic patients than in normal individuals (Roger and Walker,2005).

Acid and pepsin

Powerful digestive fluids are alleged to contribute to the formation of ulcers. The stomach can protect itself from these fluids in several ways. These are produced as lubricant like mucus that coats the stomach and shields stomach tissues. They produce bicarbonate that neutralizes digestive fluids and breaks them down into less harmful substances.

NSAIDs

The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase1, which is essential for the production of prostaglandins. NSAIDs can make the stomach vulnerable to the harmful effects of acid and pepsin by interfering with the stomach‘s ability to produce mucus and bicarbonate.

Smoking

Tobacco smoking leads to atherosclerosis and vascular spasms, causing vascular insufficiency and promoting the development of ulcers through ischemia. Nicotine contained in cigarettes can increase parasympathetic nerve activity to the GIT by acting on the nicotinic receptors at synapses– increase stimulation to the enterochromaffin like cells and G cells increases the amount of histamine and gastrin secreted, and therefore increases acidity of gastric juice (Tiwari, 2005).

Caffeine

Beverages and foods that contain caffeine can stimulate acid secretion in the stomach. This aggravates an existing ulcer, but the stimulation of stomach acid cannot be attributed solely to caffeine.

Alcohol

Heavy consumption of alcohol causes liver cirrhosis. Ulcers are found in people with liver cirrhosis.

Stress

Emotional stress do not cause ulcers, but people who are experiencing this often report increased pain in existing

ulcers. Physical stress cause increase in risk of developing ulcers, especially in stomach. Examples of physical stress that can lead to ulcers are that suffered by people with injuries such as severe burns and people undergoing major surgery (Berroteran, 2002; Medina, 2010; Friedman,1998).

MECHANISM INVOLVED IN ULCERATION

Peptic ulcers are produced by an imbalance between the gastro duodenal mucosal defense mechanisms and damaging forces. Gastric acid and pepsin are requisite for all peptic ulcerations. Hyperacidity is not a prerequisite because only a minority of patients with duodenal ulcers has hyperacidity and is even less common in those with gastric ulcers (dyspepsy.com, 2011). Many bacteria have been found in the mucus, which was continuously secreted by mucus cells and removed on the luminal side. *H. pylori* is a bacterium, has flagella and moves through the stomach lumen and drills into the mucoid lining of the stomach. To avoid entry into the lumen, *H. pylori* senses the pH gradient within the mucus layer by chemotaxis and swims away from the acidic contents of the lumen towards the more neutral pH environment of the epithelial cell surface. *H. pylori* is also found on the inner surface of the stomach epithelial cells and occasionally inside epithelial cells. It produces cytotoxin associated gene A proteins (cag A) and vacuolating cytotoxins such as vacA, which activate the inflammatory cascade. *H. pylori* expresses sialic acid specific hemagluttinins and a lipid binding adhesins that mediate the binding to the mucosal surface. Gastrin is the main hormone involved in stimulating gastric acid secretion, and gastrin homeostasis is altered in *H. pylori* infection. The hyper acidity in duodenal ulcer may result from *H. pylori* induced hypergastrinemia. The elevation of gastrin may be a consequence of bacterial mediated decrease of antral D cells that secrete somastatin, thus using the inhibitory modulation of somatostatin on gastrin, or direct stimulation of gastrin cells by certain cytokines liberated during the inflammatory process. The organisms also elaborate phospholipases which damage surface epithelial cells and may release bioactive leukotrienes andeicosanoids. *H. pylori* produce large amounts of enzyme urease, molecules of which are localized inside and outside of the bacterium. Urease breakdown urea (which is normally secreted into the stomach) to carbondioxide and ammonia (ammonia is converted into the ammonium ion by taking hydrogen from water upon its breakdown into hydrogen and hydroxyl ions. Hydroxyl ions then react with carbondioxide, producing bicarbonate which neutralizes gastric acid. The survival of *H. pylori* in the acidic stomach is dependent on urease, and it would eventually die without the enzyme. The ammonia that is produced is toxic to the epithelial cells. Neutrophils attracted by *H. pylori* release myeloperoxidase which produces hypochlorous acid yield, in turn, monochloramine can destroy mammalian cells. In addition to *H. pylori* elaboration of enzymes, other antigens recruit inflammatory cells to the mucosa. The chronically inflamed mucosa is more susceptible to the acid injury. Finally, damaged mucosa is thought to permit leakage of tissue nutrients into the surface microenvironment thereby sustaining the bacillus. Mechanisms by which *H. pylori* could promote cancer are under investigation. One mechanism involves the enhanced production of free radicals near *H. pylori* and an increased rate of host cells mutation. The mechanism has been called “perigenetic pathway” and involves enhancement of the transformed host cell phenotype by means of alterations in cell proteins such as adhesion proteins. It has been proposed that *H. pylori* induces inflammation and locally high levels of TNF-α and/or interleukin 6. According to the proposed perigenetic mechanism, inflammation associated signaling molecules such as TNF-α can alter gastric epithelial cells adhesion and lead to the dispersion and migration of mutated epithelial cells without the need for additional mutations in tumor suppressor genes such as genes that code for cell adhesion proteins.

Complications associated with PUD

Gastrointestinal bleeding

Abrupt large bleeding which is life threatening can occur when the ulcer erodes one of the blood vessels.

Penetration

Herem the ulcer continues into the adjacent organs such as liver and pancreas.

Scarring and swelling

Scarring and swelling due to ulcers causes narrowing in the duodenum and gastric outlet obstruction, which causes severe vomiting.

Pyloric stenosis

Zollinger-Ellison syndrome

It is a rare syndrome which consists of a triad of non-beta islet cell tumors of the pancreas that contain and release gastrin, gastric acid hyper secretion and severe ulcer disease. Extra pancreatic gastrinomas are also common and may be found in the duodenal wall (Robbins and Cotran, 2006).

Diagnosis

An esophago gastro duodenoscopy (EGD) is a form of endoscopy, also known as gastroscopy, is carried out on patients in whom a peptic ulcer is suspected (Humphrey et al., 2008). The diagnosis of *H. pylori* can be made by: Urea breath test (non invasive and does not require EGD), direct culture from an EGD biopsy specimen, direct detection of urease activity in a biopsy specimen by rapid urease test, stool antigen test, histological examination and staining of an EGD biopsy.

If a peptic ulcer perforates, air will leak from inside the gastrointestinal tract which always contains some air into the peritoneal cavity which never contain air. This in turn lead to "free gas" within the peritoneal cavity. If the patient stands erect, while taking a chest X-ray, the gas will float to a point beneath the diaphragm. Thus in the peritoneal cavity, an erect chest X-ray or supine lateral abdominal X-ray will be obtained which is an open or perforated peptic ulcer disease.

Antiulcer drugs (Chaudri, 1991)

Drugs which neutralize gastric acid (antacids) are: Systemic antacids eg:- sodium bicarbonate; non systemic antacids; buffer type Eg: aluminium trioxide; non buffer type Eg:- MgO, magnesium hydroxide; Miscellaneous Eg: Alginates; drugs which reduce gastric acid secretion; H_2 receptor antagonists Eg: Cimetidine; Proton pump inhibitors Eg: omeprazole; anticholinergics Eg: propantheline; prostaglandin analogs Eg:- Misoprostol; mucosal protective drugs Eg: sucralfate; ulcer healing drugs Eg: carbenoxolone; anti *H. pylori* drugs Eg:- tetracycline, amoxicillin.

Side effects of antacids (Dharmani, 2006)

Osteomalacia, chronic renal failure, belching, flatulence, feeling of fullness, nausea, exacerbation of esophageal reflux.

Side effects of anti secretory agents

Rashes, diarrhoea, muscle pain, fatigue, bradycardia, blockade of cerebral H_2 receptors can cause drowsiness, mental confusion, delirium, hallucination. On long term use, it causes hepatotoxicity, gynecomastia, hyper prolactinemia.

Mechanism of proton pump inhibitors (PPI's)

PPI's decrease basal and stimulated gastric acid secretion during inhibition of acid secretion by parietal cells, the H^+/K^+ ATPase proton pump. These agents are most effective anti secretory agents. All medications in this class are week bases that must be activated by acid to inhibit the proton pump. Paradoxically, these prodrugs are acid labile compounds that can be degraded by stomach acid during oral administration and therefore available as enteric coated delayed release formulations. Once the drug reaches the higher pH of the duodenum, the enteric coating dissolves and the unprotanated prodrug readily penetrates the cell membranes, specifically that of parietal cells. As it traverses the parietal cell, and is exposed to intracellular acid, the prodrug becomes protonated and is no longer able to freely cross the cell membranes, thus the activated PPI becomes trapped in the parietal cell. Once formed, the active sulphonamide moiety covalently binds to H^+/K^+ ATPase and inhibits acid secretion. Food may delay the absorption of some agents, but because PPI's require accumulation and acid activation, and because they inhibit only proton pumps that are actively secreting acid, they are most effective when taken on an empty stomach, shortly before meals (Satoskar, 2007).

TREATMENT OF *H. PYLORI* INFECTION

Once *H. pylori* is detected in patients with peptic ulcer, it has to be eradicated and the ulcer is allowed to heal. The standard first line therapy is a one week triple therapy consisting of proton pump inhibitor such as omeprazole and antibiotics like clarithromycin amoxycillin.

An increasing number of infected individuals are found to wharf antibiotic-resistant bacteria. This results in initial treatment failure and requires additional rounds of antibiotic therapy or alternative strategies such as a quadruple therapy (Table 1), which adds a bismuth colloid. For the treatment of clarithromycin-resistant strains of *H. pylori* use of levofloxacin as part of therapy is optional (Tiwari, 2005).

Prevention

plenty of vegetables rich in beta carotene, fruit containing vitamin C, zinc rich foods such as whole grains and sea food (oysters) should be eaten. Eating more vegetables and fruit such as carrots, kale, red and green peppers, citrus fruits, apricots, kiwi fruit may promote healing of peptic ulcers and protect against further damage to the gut wall. The helpful nutrients in these foods are β-carotene, which the body converts to vitamin A and C. Foods rich in zinc such as whole grains and seafood, can also help in the healing process. Essential fatty acids (found in fish oils and seed oils) may help to protect against ulcers by increasing the production of prostaglandlins (a group of compounds, one function of which is protect the lining of the alimentary canal).

Table 1. Triple and quadruple regimen for treatment of *H. pylori* induced ulcers.

Regimen	Duration (days)	Efficacy
Amoxicillin + PPI	14	<70-80
Clarithromycin + PPI	14	>70-90
Clarithromycin + RBC	14	>70-90
Clarithromycin + Amoxicillin+ PPI	10-14	>80-90
Clarithromycin + Metranidazole+ PPI	10-14	>80-90
Clarithromycin + Tetracycline+ PPI	14	>80-90
Tetracycline + Metranidazole+ BSS+ PPI	7-10	>80-90
Tetracycline + Metranidazole +BSS+H_2RA	14 days	>80-90
Clarithromycin+Metranidazole+BSS+PPI	7-10 days	> 80-90

BSS: Bismuth subsalicylate; RBC: ranitidine bismuth citrate; PPI: proton pump inhibitor; H_2RA: histamine H_2 receptor antagonist.

Table 2. List of few medicinal plants scientifically proven for anti ulcer activity (Dharmani, 2006; Sandhya, 2010).

Botanical name	Plant part	Extract type	Ulcer model
Terminalia pallida	Leaves	Ethanol	Indomethacin, histamine, Alcohol
Allophylus serratus	Leaves	Ethanol	Aspirine, pylorus ligated, alcohol, cold resistant
Alpinia galangal	Rhizome	Ethanol	stress, pylorus ligated, ethanol,HCl
Anchusa strigosa	Root	Aqueous	Ethanol
Artemisia herba-alba	Leaves	Aqueous	Ethanol
Astronium urundeuva	Bark	Aqueous	Aspirin,stress,histamine
Atractyloids lancea	Rhizome	Acetone	Ethanol, HCl
Azadirachta indica	Leaves	Aqueous	Stress, ethanol
Baccharis triptera	Small branches	Aqueous	Pylorus ligated, stress, indomethacin
Bauhinia racemosa	Flower buds	Methanol	Aspirin
Bryophyllum pinnatum	Leaves	Methanol	Aspirin, indomethacin, serotonin, reserpine, stress, ethanol,
Caesalpinia ferrea	Stem	Crude	Acetic acid
Camellia sinensis	Leaves	Aqueous	Stress, ethanol, aspirin, indomethacin, reserpine, histamine, serotonin
Cassia nigrans	Leaves	Ethanol	Aspirin, pylorus ligated
Cistus incanus	Aerial part	Aqueous	HCl, ethanol, reserpine, serotonin
Curcuma longa	Rhizome	Ethanol	Pylorus ligated, cold-restraint, stress, indomethacin, reserpine, ethanol
Diodia sarmentosa	Whole plant	Ethanol	Aspirin, pylorus ligated
Entandrophragma utile	Bark	Aqueous	Ethanol,
Eremomastax speciosa	Leaves	Aqueous	Ethanol, HCl, pylorus ligated
Ficus exasperata	Leaves	Ethanol	Aspirin, pylorus-ligated
Laurus nobilis	Seeds	Ethanol	Ethanol
Maytenus aquifolium	Leaves	Aqueous	Indomethacin, cold-restraint stress
Microgramma squamulosa	Rhizome	Crude,ethanol, water	Stress, ethanol, HCl, acetic acid
Mikania cordata	Root	Methanol	Stress, ethanol, aspirin, phenyl butazone, pylorus ligated
Moringa pterygosperma	Flower buds	Methanol	Aspirin
Pistacia lentiscus	Resin from stem		Pylorus ligated, aspirin, reserpine restraint plus cold stress
Pluchea indica	Root	Methanol	Indomethacin, ethanol, aspirin
Punica granatum	Fruit peel	Aqueous	Ethanol
Pyrenacantha staudtii	Leaves	Aqueous	Aspirin, indomethacin, reserpine
Quercus ilex	Root bark	Aqueous	ethanol,
Saussurea lappa	Root	Acetone	Stress

Table 2. Cont.

Stachytarpheta cayennensis	Whole plant	Aqueous	Stress, ethanol, pylorus ligated
Stryphnodendron adstringens	Aerial parts	Total extract	Stress,ethanol, indomethacin
Styrax camporum	Stem	Ethyl acetate	Acetic acidl
Swertia chirata	Whole plant	Ethanol	Indomethacin, pylorus ligated, ethanol
Synclisia scabrida	Leaves	Ethanol	Aspirin, pylorus ligated
Tanacetum vulgare	Aerial parts	Chloroform	Ethanol
Trianthema pentandra	Whole plant	Methanol	Aspirin
Trichosanthes kirilowii	Fruit	Ethanol	Stress, histamine, serotonin, ethanol, HCl
Vernonia kotschyana	Root	Aqueous	Pylorus ligated, stress, indomethacin
Zingiber officinalis	Root	Methanol, acetone	HCl/ethanol
Amphipterygium adstringens	Stem bark	Methanol	Ethanol
Desmodium gangeticum	Root	Ethanol	Aspirin, alcohol, pylorus ligated, cold resistant
Ocimum sanctum	Leaves	Ethanol	Aspirin, alcohol, pylorus ligated, cold resistant,histamine
Hemidesmus indicus	Not mentioned	Ethanol	Aspirin, pylorus ligated
Asparagus racemosa	Fresh roots	Fresh juice	Aspirin, alcohol, pylorus ligated, cold resistant,histamine,cold resistant,cysteamine
Embelica officinalis	Fruits	Methanol	Aspirin, alcohol, pylorus ligated, cold resistant
Bacopa monniera	Not mentioned	Fresh juice	Aspirin, alcohol, pylorus ligated, cold resistant
Bidens pilosa	Not mentioned	Ethanol	Alcohol, pylorus ligated, indomethacin
Musa sapientum	Not mentioned	Powder	Pylorus ligated
Polyscias balfouriana	Leaves and root	n-butanol	Aspirin and physical stress induced

Intake of salt, soya sauce, spicy foods, caffeine in coffee, tea, cola drinks and alcohol should be cut down on. Large meals should be avoided, as they can encourage the production of excessive acid. Sufferers may also find that chilli peppers, black pepper, mustard and other strong spices such as those found in curries may aggravate their symptoms (Reader's Digest, 1996).

Medicinal plants as potent anti ulcer agents

Medicinal plants form the backbone of traditional systems of medicine in India. Phytochemicals from medicinal plants serve as lead compounds in drug discovery and design. Medicinal plants are rich source of novel drugs that forms the ingredients in traditional systems of medicine, modern medicines, nutraceuticals, food supplements, folk medicines, pharmaceutical intermediates, bioactive principles and lead compounds in synthetic drugs. WHO pointed out that more than 80% of world's population depends on plants to meet their primary health care need. India is one of the 12 mega diversity countries in the world so it has a vital stake in conservation and sustainable utilization of its biodiversity resources. Plant extracts are some of the most attractive sources of new drugs and have been shown to produce promising results for the treatment of gastric ulcer. Nearly 240 medicinal plants and 21 plants based compounds were identified as anti ulcer agents so far (shodhganga.inflibnet.ac.in, 2011) (Table 2 and 3).

Conclusion

Medical treatments are effectual for some people, but not for everyone. They cause many superfluous side effects and make symptoms worse. As doctors continue to prescribe the same antibiotics, *H. pylori* resistance will continue to increase and the medications will become less effective. For minimizing the side effects, potent herbal drugs which can eradicate all traces of *H. pylori* is the better choice.

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Table 3. List of medicinal plants used by the folklore of Andhra Pradesh, India for ulcer treatment (Madhava et al., 2008).

Botanical name	Family	Parts used
Abutilon indicum	*Malvaceae*	Leaf
Acacia nilotica	*Mimosaceae*	fruit, seed, gum,resins
Albizia amara	*Mimosaceae*	Flower
Allium sativum	*Alliaceae*	Bulb
Ammannia baccifera	*Lythraceae*	Whole plant, leaf
Ampelocissus latifolia	*Vitaceae*	Leaf
Anacardium occidentale	*Anacardiaceae*	Rootbark
Antidesma ghaesembilla	*Stilaginaceae*	Leaf
Asphodelus tenuifolius	*Liliaceae*	Seed
Azadirachta indica	*Meliaceae*	Leaf
Balanites aegyptiaca	*Belanitaceae*	Leaf
Bambusa arundinacea	*Poaceae*	Root, leaf, fruit ,seed
Bauhinia variegate	*Caesalpiniaceae*	Stem bark
Boswellia ovalifoliolata	*Burseraceae*	Stem
Boswellia serrata	*Burseraceae*	Gum
Caesalpinia coriaria	*Caesalpiniaceae*	Fruit
Calophyllum inophyllum	*Clusiaceae*	Stembark
Calycopteris floribunda	*Combretaceae*	Leaf
Canavalia gladiate	*Fabaceae*	Pod
Canna indica	*Cannaceae*	Root
Carallia brachiata	*Rhizophoraceae*	Fruit
Cassia absus	*Caesalpiniaceae*	Leaf
Celosia argentea	*Amaranthaceae*	Whole plant
Clitoria ternatea	*Fabaceae*	Root
Combretum albidum	*Combretaceae*	Leaf
Coriandrum sativum	*Apiaceae*	Fruit
Cucurbita moschata	*cucurbitaceae*	Fruit seed, seed oil
Curcuma longa	*zingiberaceae*	Rhizome
Curcuma neilgherrensis	*zingiberaceae*	Rhizome
Cyclea peltata	*Menispermaceae*	Root
Dactyloctenium aegyptium	*Poaceae*	Fruit
Dalbergia latifolia	*Fabaceae*	Root
Dioscorea oppositifolia	*Dioscoreaceae*	Tuber
Dioscorea bulbifera	*Dioscoreaceae*	Tuber
Dioscorea hispida	*Dioscoreaceae*	Tuber
Dioscorea pentaphylla	*Dioscoreaceae*	Tuber
Dioscorea tomentosa	*Dioscoreaceae*	Tuber
Ficus benghalensis	*Moraceae*	Stem bark
Ficus benjamina	*Moraceae*	Leaf
Ficus religiosa	*Moraceae*	Stem bark
Ficus virens	*Moraceae*	Stem bark
Gardenia gummifera	*Rubiaceae*	Gum
Glinus oppositifolius	*Molluginaceae*	Whole plant
Gloriosa superba	*Cochlaceae*	Tuber
Hedera helix	*Araliaceae*	Leaf
Heliotropium indicum	*Boraginaceae*	Whole plant
Homonoia riparia	*Euphorbiaceae*	Root
Hydrolea zeylanica	*Hydroleaceae*	Leaf
Ipomoea eriocarpa	*Convolvulaceae*	Whole plant
Ixora coccinia	*Rubiaceae*	Flower

Table 3. Cont.

Jasminum sambac	*Oleaceae*	Leaf
Jatropha curcas	*Euphorbiaceae*	Whole plant
Jatropha gossypifolia	*Euphorbiaceae*	Latex
Kalanchoe laciniata	*Crassulaceae*	Leaf
Lablab purpureus	*Fabaceae*	Leaf
Lactuca sativa	*Asteraceae*	Latex,stem
Lannea coromandelica	*Anacardiaceae*	Leaf
Lawsonia inermis	*Lythraceae*	Stem bark, leaf, flower
Luffa acutangula	*Cucurbitaceae*	Root
Macaranga peltata	*Euphorbiaceae*	Leaf
Madhuca longifolia	*Sapotaceae*	Root bark
Mangifera indica	*Anacardiaceae*	Root bark,stembark
Momordica charantia	*Cucurbitaceae*	Fruit
Morinda pubescens	*Rubiaceae*	Root, fruit
Myrtus communis	*Myrtaceae*	Fruit
Nelumbo nucifera	*Nelumbonaceae*	Whole plant
Nymphaea nouchali	*Nymphaceae*	Whole plant
Ochna obtusata	*Ochnaceae*	Leaf
Persea macrantha	*Lauraceae*	Leaf
Phoenix sylvestris	*Areceaceae*	Root
Phyla nodiflora	*Verbenaceae*	Whole plant
Pimpinella tirupatiensis	*Apiaceae*	Tuber
Plumbago zeylanica	*Plumbaginaceae*	Leaf
Polycarpaea corymbosa	*Caryophyllaceae*	Whole plant
Pongamia pinnata	*Fabaceae*	Root
Portulaca oleracea	*Portulacaceae*	Whole plant
Portulaca quadrifida	*Portulacaceae*	Whole pant
Pouzolzia wightii	*Urticaceae*	Leaf
Pouzolzia zeylanica	*Urticaceae*	Leaf
Pterocarpus santalinus	*Fabaceae*	Heartwood
Rosa centifolia	*Rosaceae*	Flower
Schleichera oleosa	*Sapindaceae*	Stem bark
Sesbania sesban	*Fabaceae*	Flower
Shorea tumbugggaia	*Dipterocarpaceae*	Resin
Sigesbeckia orientalis	*Asteraceae*	Whole plant
Solanum giganteum	*Solanaceae*	Leaf
Solanum melongena	*Solanaceae*	Root
Solidago virga aurea	*Asteraceae*	Whole plant
Sorghum vulgare	*Poaceae*	Fruit
Stachytarpheta jamaicensis	*Verbenaceae*	Whole plant
Syzygium alternifolium	*Myrtaceae*	Fruit
Talinum portulacifolium	*Portulacaceae*	Leaf
Tamarindus indica	*Caesalpiniaceae*	Leaf
Tectona grandis	*Verbenaceae*	Heartwood
Tephrosia calophylla	*Fabaceae*	Leaf, tuberous root
Tephrosia maxima	*Fabaceae*	Whole plant
Tephrosia purpurea	*Fabaceae*	Whole plant
Terminalia arjuna	*Combretaceae*	Fruit
Terminalia pallida	*Combretaceae*	Fruit
Trigonella foenum-graecum	*Fabaceae*	Seed
Triumfetta rhomboidea	*Tiliaceae*	Root
Tylophora fasciculata	*Asclepiadaceae*	Leaf

Table 3. Cont.

Viscum articulatum	*Viscaceae*	Whole plant
Wrightia tinctoria	*Apocyanaceae*	Latex
Xanthium indicum	*Asteraceae*	Leaf
Yucca gloriosa	*Agavaceae*	Whole plant

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